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## Tissue factor coagulation pathway and blood cells activation state in renal insufficiency.

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**Mercier E, Branger B, Vecina F, Al-Sabadani B, Berlan J, Dai M, Fourcade J, Gris JC.**

Laboratoire d'Hematologie, Centre Hospitalier Universitaire, Nîmes France.

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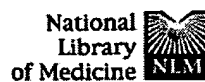
**INTRODUCTION:** Atherosclerotic cardiovascular disease is the leading cause of the increased morbidity and mortality observed in uremic patients. Thrombosis is an important contributor to the evolution of atherosclerotic lesions. The physiologically-relevant blood clotting depends on binding of activated factor VII (FVIIa) to exposed tissue factor (TF) on activated/damaged cells. **MATERIALS AND METHODS:** A cross-sectional study was performed on three age-sex-matched groups of individuals: one group of 50 patients on maintenance hemodialysis (D group), one of 50 patients with a non-dialysed renal insufficiency (ND group) and one of 50 healthy controls (HC group). We studied basal plasma concentrations of F factor VII-related antigen (FVIIAg), soluble TF, tissue factor pathway inhibitor (TFPI), TF-dependent circulating monocytes procoagulant activity (TF-dMPA), tissue factor-dependent plasma reactivity to activated protein C (TF-aPC), D-dimers (D-Di), and circulating markers of cellular activation/injury: soluble thrombomodulin (sTM), circulating microparticles (microP), soluble leukocyte, endothelial and platelet selectins (sL-selectin, sE-selectin, sP-selectin), soluble intercellular adhesion molecule 1 and vascular cell adhesion molecule 1 (sICAM-1 and sVCAM-1). Their variations induced, in hemodialysis patients during dialysis run were thereafter studied. **RESULTS:** Values of FVIIa, FVIIa/FVIIAg ratio, sTF, TFPI, TF-dMPA, D-Di, sTM, microP, sL and sP selectins, sICAM-1 and sVCAM-1 increased all along the hierarchy HC group/ND group/D group. Microparticles were mainly of platelet origin, to a lesser extent of monocyte origin. Dialysis indu-

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## **In vivo competition between a metallothionein regulatory element and the SV40 enhancer.**

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The human metallothionein-IIA (hMT-IIA) gene contains an enhancer element within its 5' regulatory region. This enhancer element can compete with the SV40 enhancer for one or more cellular factors *in vivo*. The competition between the two elements is modulated by cadmium, an inducer of hMT-IIA transcription. The data presented are consistent with a model in which heavy metal ions control the ability of the hMT-IIA enhancer to bind a positive factor, leading to increased transcription. The same factor is required for maximal activity of the SV40 enhancer, which suggests that viruses utilize factors that have a normal role in cellular gene expression to control their own genes.

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